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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

PUBLISHED MONTHLY for the PHYSICIAN in the GENERAL PRACTICE of MEDICINE

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

EDITORIAL COMMENT

Summing Up The first issue of this journal, *Diseases of the Nervous System*, was published in January, 1940. The appearance of this, the twelfth issue, marks the end of the first year of publication of a journal whose purposes and aims were set forth by the editors in an editorial of the first, or January number. Although there was a recognized need for a medical journal of this type, the editorial staff undertook publication with a feeling of trepidation.

We are pleased with the way the journal has been received by everyone, and its success thus far has removed our original doubts.

The early policy of publishing a practical journal on neurology, psychiatry, mental hygiene, and neurosurgery has been followed in all issues. Each number has contained articles on those specified medical subjects that have some interest for the physician in general practice, which, of course, is the primary aim of the journal. In addition, a very brief bibliography of the author precedes each article to aid the reader in evaluating the writer's qualifications, as well as to introduce the specialists in the field of nervous and mental diseases to the practicing physicians.

The editors wish to thank those who have contributed the articles published during this year, and those who have submitted papers for publication during the coming year. It is the wish of the editorial staff that the articles submitted be on subjects of practical interest to the entire profession, and expressed in terms or words that are in common usage. It is to be hoped that an improvement in the character of the articles in this respect will be achieved for this is the only way that the purpose of the journal will be fulfilled.

The growing circulation of the journal shows that it is being read by many outside of our own specialty. We are particularly grateful to the many neurologists, neurosurgeons and psychiatrists who have expressed the opinion that the journal has made a good start, and have offered their continued support. We hope to retain that opinion and feel that it can be accomplished through adherence to our policy of publishing only selected material.

Also, in the future an editorial on some practical phase of nervous or mental disorders that are of importance to all will appear each month. In this way we hope to clarify debatable points and present accurate information on changing opinions or new concepts of the diseases of the nervous system.

The editorial staff is taking this opportunity to thank you for your past consideration, and to tell you that we will welcome letters containing criticism and suggestions from those, other than specialists in our own field, who have followed the journal through the first year.

TITUS H. HARRIS, M.D.
Editor.

The Early Recognition of Tumors of the Brain*

PAUL C. BUCY, M.D.
Chicago, Illinois

Dr. Bucy received his Doctorate of Medicine from the State University of Iowa College of Medicine in 1927. He is, at present, Associate Professor of Surgery, in charge of neurological surgery at the University of Chicago. Dr. Bucy is a member of the American Board of Neurological Surgery, a Diplomate of the American Board of Neurology and Psychiatry and is a member of the American Neurological Association and the Society of Neurological Surgeons.

Although recent years have seen an increasing awareness of the prevalence of tumors that involve the brain, such tumors still are all too frequently not recognized. In a day when all such tumors presented a uniformly hopeless outlook this may have been a matter of little importance. But that day has passed. In a recent report, Cairns has shown that of the patients suffering from brain tumors 40 per cent were alive eight years after operation and 23.5 per cent were living "useful" lives. This represents a tremendous advance in the treatment of this condition. It is our obligation to see that every sufferer from an intracranial tumor be given every opportunity for relief or cure that modern neurological surgery offers. Nor are such sufferers rare. It has been reliably demonstrated that one or two persons out of every one hundred will suffer from such a tumor in the course of his lifetime. Generously estimating the average length of life as sixty years, one learns that in the state of Illinois, with a population of eight million people, there will be between 1,300 and 2,700 cases of brain tumor *each year*. It is obvious that if these people are to receive the greatest benefits possible their condition must be recognized early before vision has been lost and life hopelessly endangered. It is obvious that such recognition must be at the hands of the patient's regular medical adviser; though accurate localization of the tumor and appropriate treatment may be the domain of the neurologist and neurosurgeon.

There are certain common manifestations of intracranial tumors. Although these are by no means always present, nor necessarily all present in a given case, their frequency is such that their presence should always suggest the possibility of such a tumor.

Progressive Loss of Function

The most significant manifestation of a brain tumor is a *progressive loss of neurological function*. There are few other pathological states that give rise to an ever increasing impairment of some one area of the nervous system. Thus a history of unilateral tinnitus, a progressive loss of hearing in the same ear, momentary twinges of pain, numbness and tingling in the check on the same side, beginning awkwardness in the movements of the ipsilateral arm and leg, associated with pain and tenderness back of the mastoid process on that side leave little doubt that a neurinoma of the acoustic nerve is present, even though the "cardinal" signs, the "diagnostic triad" of brain tumor, headache, vomiting and failing vision have not yet put in their appearance. The following case illustrates the progressive neural deficit produced by a tumor in quite a different location.

Case I

M. A., a housewife, fifty-six years of age, was admitted to the University of Chicago Clinics on January 9, 1929 in a stuporous condition. According to her family she had been perfectly well until May, 1928. At that time she began to complain of attacks of tingling involving the back of the left hand. In a short time the numbness extended, involving the entire upper extremity, although the attacks invariably began on the dorsum of the hand. A diagnosis of "neuritis" was made. A month after the onset the left hand became weak and she frequently dropped dishes from it. Furthermore, the attacks which previously were only sensory in nature were now associated with clonic movements of the extremity. The weakness grew steadily greater

* From the Division of Neurology and Neurosurgery of the University of Chicago. Read before the La Crosse County Medical Society, La Crosse, Wisconsin, May 7, 1940.

and by October the left arm and hand were quite useless. During the first six months of the illness the attacks of numbness and clonic movements were limited to the left upper extremity, but on November 7th they extended to involve the entire left side of the body. She never lost consciousness in these attacks. Gradually the left lower extremity grew weaker and the left side of the face began to droop. Early in December walking became impossible. At that time she was seen by another physician who made a diagnosis of "cerebral embolism" in spite of this history of a steadily progressive loss of nervous function and of sensory and motor fits which were first localized and later were of the typical "Jacksonian" type. He specifically stated that the patient could not be suffering from a brain tumor because there was no choking of the optic discs and the patient had not suffered from headache nor vomited.

Her condition grew steadily worse and vision began to fail. Early in January of 1929 her condition suddenly became critical. Dr. George W. Hall of Chicago was then called to see her for the first time. He immediately recognized that a tumor of the brain was responsible for her condition and referred her to us for further care.

She was admitted on January 9, 1929 in a semi-comatose state. Examination revealed bilateral choked discs, a left lower facial weakness and a left spastic hemiplegia. A satisfactory sensory examination was not possible. Fever was present and examination of the chest revealed a pneumonia. Surgical intervention was out of the question. She grew rapidly worse and soon died of a bronchopneumonia.

Necropsy revealed a benign meningioma in the right central area. Had its presence been recognized before the terminal pneumonia developed, it could undoubtedly have been readily removed and this unfortunate woman been restored to health.

Discussion

This story presents with crystal clarity the entire progress of this tumor. It began near the sensory "hand" area of the post-central gyrus. Irritation of this focus resulted in the sensory attacks on the dorsum of the hand. Growth, however, soon caused irritation of the neighboring pre-central motor cortex. This was followed shortly by paralysis which gradually extended upward to the leg and downward to the face. Nothing except a tumor is likely to have produced this picture. Certainly neither embolism nor any other vascular process could have done so. The absence of the signs of increased intracranial pressure is of no significance other than indicating the probable benign and enucleable character of the lesion.

Convulsions

The above case also presents us with another of the common manifestations of intracranial tumors—fits. Frequently, these paroxysmal attacks are motor in character, but they may be sensory (as in case I) or visual or "so-called" uncinate seizures.

The latter are peculiar attacks in which the individual first smells a peculiar odor or experiences a peculiar taste which he is often unable to identify. He then passes into a mental state commonly referred to as "dreamy," but which the patient often describes as "being in a haze." This may terminate the attack, or there may be visual and auditory hallucinations or a generalized convulsion may develop. Such attacks are typical of a tumor in one or the other temporal lobes.

Visual attacks are less common, but the following history is typical of such seizures and illustrative of the favorable results which can be expected with brain tumors of this type.

Case II

M. D., a young lady twenty-five years of age, was referred to the University of Chicago Clinics by Dr. Maurice M. Hoeltgen of Chicago.

She had been well until the age of fifteen when she began to suffer from attacks of flickering colored lights before her eyes. These lights never seemed more to one side than the other. The spells lasted about ten minutes and occurred at intervals of a few months. They were not associated with any other symptoms and continued unchanged for nine years, when in the spring of 1936 the attacks of flickering lights began to be followed by headaches. The headaches increased in severity and duration, often per-

sisted for several hours. They were soon associated with nausea and vomiting. The frequency of the seizures increased and at the time of admission they were occurring every three of four days. The attacks usually occurred immediately after arising in the morning.

She was admitted to the Clinics on September 27, 1937. Examination revealed no abnormalities on general physical examination. Neurological examination was likewise negative except for choking of both optic discs. The visual fields were full when examined on both the Bjerrum screen and the perimeter and the central fields were full on campimetric examination. The blind spots were moderately enlarged.

On September 30th a ventriculogram was attempted, but the needle entered a cystic cavity in the left occipital lobe and 15 cc. of lemon colored fluid was removed and the cyst filled with air. Roentgenograms demonstrated the cystic tumor in the left occipital lobe.

On October 23, 1937 the left occipital lobe including the tumor, a mixed astrocytoma and spongioblastoma, was amputated. Since then she has remained perfectly well except for a persistent right homonymous hemianopia.

Discussion

The visual hallucinations were undoubtedly indicative of irritation of the visual cortex on the medial surface of the occipital lobe. It was rather surprising that the visual fields showed no defect; but examination of the amputated lobe revealed that the area striata had escaped destruction by the tumor.

Convulsive seizures are amongst the most prevalent symptoms of neurological disease. For the convenience of our consideration such seizures may be divided into localized and generalized attacks.

Localized attacks, whether they involve a small segment such as one side of the face, an arm or a leg or an entire half of the body, are indicative of localized cerebral pathology such as a scar, an abscess or a tumor. But whereas a cerebral scar is productive of a more or less stationary picture and usually arises from readily demonstrable causes such as severe trauma, surgical intervention or a cerebral infarct, tumors result in a progressive state as illustrated in Case I. Abscesses likewise present a gradually increasing symptomatology. Occasionally, the differentiation of abscess and tumor is difficult. But the presence of a localized area of intracerebral suppuration is usually clearly indicated by the presence of a primary source of infection such as an otitis media, a pulmonary abscess, etc., some evidence of infection, signs of meningeal irritation and a more rapidly progressive course.

Attacks which begin in a localized area but spread to the *entire* body commonly result from demonstrable localized cerebral pathology just as the strictly localized attacks do; but occasionally no such localized pathology is demonstrable and such cases must either be attributed to generalized cerebral changes or be classified as idiopathic.

Generalized convulsive seizures offer a more difficult diagnostic problem than do the localized attacks. On the basis of demonstrable pathology, generalized convulsions are readily divisible into two groups, those occurring in the first two decades of life and those occurring after thirty years of age. Those appearing between the ages of twenty and thirty form a borderline group.

Generalized convulsions occurring in children and adolescents are usually to be classified either as idiopathic, or as the result of diffuse cerebral disease. That such is not invariably the case has been shown by Bailey, Buchanan and Bucy in their recent book *Intracranial Tumors of Infancy and Childhood.* Generalized convulsions are common manifestations of tumors in and about the third ventricle in this age group. However, such tumors account for but a very minute percentage of all of the convulsions occurring in children. Nevertheless, their occurrence is a warning that all children subject to such attacks should be continuously observed for the development of any other evidence of an intracranial tumor.

Generalized convulsions which appear for the first time in individuals over thirty years of age present an entirely different problem. The most common causes of such convulsions are tumor of the brain, syphilis and cerebral arteriosclerosis. *Syphilis* is rather readily excluded. However, it should be pointed out that

its presence by no means establishes it as the cause of the symptoms and excludes tumor. In any case where the Wassermann test is positive, but the symptoms are those of an expanding localized intracranial lesion the patient should be treated as the victim of a tumor until that possibility has been excluded. Unfortunately, the treponema pallidum does not immunize its victims against neoplasms.

The differentiation of generalized convulsions caused by tumor from those resulting from arteriosclerosis is often difficult. However, the age incidence of the two furnishes a very helpful basis for the differentiation of many of these cases. Whereas brain tumors during the adult years are most prevalent between the ages of thirty and fifty with the peak of incidence at forty-five years, cerebral arteriosclerosis most commonly develops after fifty and the percentage of its victims increases with every year thereafter.

A few other rarer conditions should also be considered whenever convulsions develop in an adult. The more common are hyperinsulinism, hypersensitivity of the carotid sinus reflex, and cerebral degenerative states such as Pick's and Alzheimer's diseases.

The following case is typical of many of those in which generalized convulsion develops as a result of a cerebral tumor.

Case III

O. P., a plasterer, thirty-eight years of age, was referred to the University of Chicago Clinics by Dr. J. P. Graf of Chicago, who had been called in consultation by the family only shortly before. In 1929, six years before, he had first suffered from a loss of consciousness and a generalized convulsion. These attacks at first occurred at intervals of about six months and later occurred somewhat more frequently. In May, 1935 he began to complain of headaches which gradually increased in severity and frequency. Diplopia developed, and in July, one month before his admission, he began to vomit. Both the headaches and vomiting tended to occur in the morning immediately after breakfast. But for the two weeks before admission the headaches were almost continuous.

He was admitted to the University of Chicago Clinics at 2:00 p. m. on August 21, 1935 in a stuporous condition. General physical examination revealed no abnormality except that the pulse rate was reduced to 40 per minute. The blood pressure was 128/70 and the respiratory rate was 20. There was no choking of the optic discs. All tendon reflexes were increased and Babinski's sign was present bilaterally. No paralysis or anesthesia could be demonstrated within the limits imposed by his condition. A lumbar puncture was performed but no fluid was allowed to escape except into the small bore manometer. The pressure was 350 mm. of fluid. A ventriculogram was performed which demonstrated an expanding lesion in the right frontal lobe. However, his condition rapidly failed and he died a few hours later at 9:30 p. m. on August 22nd.

Necropsy revealed an enormous benign glioma, astrocytoma, of the right frontal lobe.

This case illustrates the fact that generalized convulsions may constitute the sole manifestation of a cerebral neoplasm for years before any other symptoms either of cerebral involvement or of increased intracranial pressure develop. There can be but little question that earlier recognition of the underlying pathology in this case would have prolonged this man's life considerably. As in case I, when the terminal stage of coma and pulmonary infection has been reached, the surgeon's hands are often tied and it becomes impossible for him to do what a few weeks or months earlier would have been eminently possible.

It is noteworthy that generalized convulsions are not produced by cerebellar tumors. As the cerebellum is the most common location of brain tumors in children, it is easy to understand why such convulsions are not commonly associated with brain tumors in this age group. However, convulsions in association with intracranial hypertension do occur in children. Such a development is usually indicative of lead poisoning with a toxic encephalopathy, a tumor in or about the third ventricle or a subdural hematoma.

Lead intoxication in children is usually readily demonstrable either by a roentgenogram of the long bones, which reveals a line of increased density at the ends of the shafts of the long bones, or by finding basophilic stippling of the red blood cells, or both. Lead

encephalopathy in children is also frequently associated with hyperextension and stiffness of the neck and an increase in the number of lymphocytes and amount of protein in the cerebrospinal fluid. Unlike adults, children with lead intoxication do not develop evidence of peripheral neuritis frequently. One of the most effective forms of treatment of lead encephalopathy in a child is a cranial decompression.

Subdural hematomas in children are most prevalent during the first two years of life and apparently result from natal trauma. Frequently, their only manifestation is the development of a hypercephalus. And any hydrocephalus that is not present at birth should be suspected as due to this cause. In other cases convulsions, vomiting, generalized debility, ocular palsies, retinal haemorrhages, papilloedema, etc., occur. The diagnosis is readily made in an infant by introducing a needle through the dura mater at the lateral angle of the anterior fontanelle. In older children, a small trephine opening in the skull in the upper temporal region will be required.

Failing Vision

Although a gradual failure of vision may result from many causes, brain tumors are amongst the most prevalent. Failure of vision in such cases usually results either from the secondary optic atrophy which succeeds unrelieved papilloedema, or from direct pressure upon the optic nerves. Blurring of vision may also develop as a result of strabismus. Such crossing of the eyes associated with brain tumors is most commonly due to a paralysis of the abducens or sixth cranial nerve. In the absence of increased intracranial pressure, particularly in a child, this is highly suggestive of a glioma of the pons. When associated with intracranial hypertension, the paralysis can usually be attributed to the pressure and is of little localizing significance.

The failing vision that results from choking of the optic discs and secondary optic atrophy is readily recognizable upon making an opthalmoscopic examination. In those cases where useful vision still remains, the visual fields usually reveal only a concentric constriction. There is little excuse in this day for any patient's ever losing his vision as the result of papilloedema. Fortunately, nature allows a considerable period between the onset of the papilloedema and the failure of vision, ample time for the condition to be recognized and relieved. Unfortunately, vision once lost from this cause is practically never regained. And with our present knowledge, such blindness is indicative of serious negligence on the part of the parents, the patient or the physician.

The following case is a most interesting one in which failing vision was the symptom which caused the patient to seek medical aid and the one which led to the correct diagnosis and treatment. It also demonstrates the results that may be obtained by modern neurosurgical methods when the pathological condition is promptly recognized.

Case IV

M. S., a housewife, thirty-three years old, was referred to the University of Chicago Clinics by Dr. C. V. Elliott and Dr. C. Clement of Chicago. In May, 1937, four months prior to admission, she became aware of slight blurring of vision. She therefore had glasses fitted by an optometrist and for a short time the condition was relieved. The blurring of vision then recurred, being most severe in the left eye. Two additional changes of glasses gave no relief. She denied having suffered from any headache or vomiting and had no other complaints. Examination by the referring physicians had revealed choking of both optic discs.

She was admitted on September 6, 1937. General physical examination revealed nothing abnormal. On neurological examination the only positive findings were bilateral papilloedema of five diopters in the left eye and four in the right with a few hemorrhages over and around the left disc. The visual acuity was 1.0 in both eyes. A variable nystagmus was present on looking to either side. The corneal reflex was diminished on the left side and there was a very slight hypalgesia over the left side of the face. Examination with the audiometer revealed a mild loss of hearing for the high tones which was equal in the two ears. But the vestibular system in the left ear did not respond to the caloric tests. Roentgenograms of the skull revealed questionable dilatation of the left internal auditory meatus and slight enlargement of

the sella turcica with thinning of the dorsum.

Diagnosis of a left acoustic neurinoma was made. At operation on September 9, 1937, a left acoustic neurinoma was found and completely removed. The facial nerve was sacrificed. She made a rapid recovery and was discharged from the hospital twelve days later on September 21st.

On November 11 a facial-spinal accessory nerve anastomosis was performed. She has continued in excellent health since.

Failure of vision as a result of direct compression of the optic nerves is most commonly the result of pituitary tumors. Although ophthalmoscopic examination in such cases often reveals primary optic atrophy such changes are frequently absent early in the course of the disease. In these cases examination of the visual fields is of great value. In cases of pituitary tumors, loss of vision in the visual fields lateral to the points of fixation, a bitemporal hemanopsia, is the most common finding. The following history is typical of such cases. Although the condition had advanced much further than is often compatible with good results from treatment this patient was very fortunate.

Case V

P. W., a housewife, forty-six years old, was referred to the University of Chicago Clinics by Dr. James P. Dean of Madison, Wisconsin.

In 1934, three years before admission, she suddenly ceased menstruating. She was then forty-three years old. Soon thereafter she noted that her hands and feet were enlarging. In 1935 she complained of a choking sensation. Examination revealed a nodular enlargement of the thyroid gland and a B.M.R. of +12. Thyroidectomy was performed on May 10, 1935. In February, 1937 she suddenly noted that she was unable to see out of the left eye and that vision was very poor in the right. She also began to suffer from generalized weakness and from mild headaches, which gradually increased in severity.

She was admitted to the University of Chicago Clinics on August 24, 1937. Her visual acuity was 0.3 in the right eye and 3/200 in the left. The temporal half of the right visual field was blind. There was a central scotoma in the left field and because of the impaired vision the outlines of the peripheral field could not be accurately determined. There was a mild bilateral primary optic atrophy. Except for the occasional elicitation of Babinski's sign on the left side the neurological examination was negative. The hands and facial features were definitely enlarged. The skin, particularly of the forearms, was coarse. The teeth were widely spaced. The B. M. R. was −1. Roentgenograms revealed a marked enlargement of the sella turcica with severe decalcification of the dorsum.

Diagnosis of a pituitary adenoma with mild acromegaly was made. At operation on August 28, 1937, a cystic adenoma of the hypophysis was found and evacuated. Within less than twenty-four hours visual acuity had begun to improve and the visual fields were filling out. She recovered rapidly and was discharged from the hospital on September 8th, eleven days after the operation.

On April 7, 1938, about eight months later, Dr. Dean reported that she was in good health. Dr. E. E. Neff and Dr. F. A. Kundert of Madison, Wisconsin reported that her visual fields were normal and that visual acuity was 20/30 in the right eye and 20/40 in the left.

Headache

Although at one time headache and the other manifestations of intracranial hypertension were looked upon as the *sine qua non* for the diagnosis of an intracranial tumor, such is no longer true. Many cases are now being recognized long before this stage is reached. In others, however, the symptoms of increased intracranial pressure are still the first manifestation of the disease. Every headache should be a suspect. However, the typical headaches produced by intracranial hypertension have certain characteristics: The pain is severe; it has a bursting or throbbing character. Almost never is the pain described as superficial, in the scalp. The headaches which are described as a pressure down upon the head or inward upon the temples are usually functional in origin. The headaches associated with brain tumor tend to recur in the same region. However, their location is frequently of no significance in indicating the site of the tumor. These headaches are often not continuous. Days may elapse in which the patient is free from pain.

At the onset, the pain tends to occur in the early morning just before or after arising. At first the headaches are of short duration and may last only for a few minutes. Gradually, the duration increases. They tend to occur at other hours of the day particularly when the patient strains at stool, lifts a heavy object, stoops over, or lies down. They may awaken the patient from his sleep in the early morning hours. Eventually, they become almost continuous and unbearable. Every patient suffering from headaches, particularly if they follow the pattern outlined above, should receive a careful ophthalmoscopic, neurologic and general physical examination.

Vomiting

The headaches of intracranial hypertension are often associated with vomiting, which on occasions may relieve the pain. Contrary to old teaching, the vomiting is seldom projectile in character. Usually it is associated with nausea and does not differ from vomiting from other causes except that there is a definite tendency for the vomiting, like the headache, to occur in the early morning on arising. Although this dissociation of vomiting from the ingestion of food is highly suggestive, it does not always hold true; the vomiting caused by a brain tumor is frequently precipitated by the taking of food. Any patient who suffers from persistent vomiting should be suspected of harboring a brain tumor and the case fully investigated. Recurrent vomiting is a particularly valuable sign in recognizing the presence of intracranial tumors in children. Because of the possibility of their skulls enlarging by separation of the sutures, headaches in these cases are often relieved or lessened by such a spontaneous decompression. Furthermore, the uncomplaining nature of small children and their inability to describe accurately a new experience also tend to make headache a less common complaint in children than in adults suffering from brain tumors. Such objective manifestations as vomiting thus achieve added value in these young patients. The physician attending children must be ever aware of the fact that vomiting is not always the result of dietary indiscretions, appendicitis or other gastro-intestinal disturbances. The records of all neurosurgical clinics contain many instances where vomiting has centered the physician's attention upon the gastro-intestinal tract to the exclusion of the nervous system. Under such circumstances, hazardous delays have resulted and, in addition, unnecessary appendectomies and tonsillectomies have been performed and other forms of useless therapy engaged in.

Conclusion

It may prove useful to reiterate briefly the major points to which attention has been called in this paper:

1. Any progressive loss of neurological function, explicable upon the basis of a single lesion within the brain is strongly suggestive of a tumor of the brain.

2. Localized or Jacksonian convulsions are indicative of localized cerebral lesions of which tumors are one of the most prevalent.

3. Generalized convulsions appearing for the first time in an adult, particularly if he be between thirty and sixty years of age, are very suggestive of the presence of a brain tumor.

4. Failure of vision should always provoke a thorough investigation and the possibility that an intracranial neoplasm is at fault should be given adequate consideration.

5. Recurrent headache and vomiting, particularly when they tend to develop first and most commonly in the early morning and to increase gradually in frequency and severity are strongly suggestive of the presence of a brain tumor.

950 East 59th Street.

MEETING

On October 3, 1940, the New Orleans Society of Neurology and Psychiatry had their first annual meeting:

Dr. Edmund Connely and Dr. Gilbert Anderson presented a case: *Narcoleptic Syndrome Favoring Encephalitis.*

Dr. Walker Thompson presented a paper entitled: *Clinical Electroencephalography.*

Denial in Mental Illnesses

GEORGE S. SPRAGUE, M.D.
White Plains, New York

Dr. Sprague graduated from Johns Hopkins Medical School in 1920. He was Assistant Physician, later Acting Assistant Director, of the Iowa State Psychopathic Hospital, and Assistant Professor of Psychiatry in the Iowa University Medical School. In 1927, he became Clinic Director of the Iowa Mobile Mental Hygiene Clinic. Following a year's study in psychoanalysis in Europe during 1931, he became physician-in-charge of the Men's Division, New York Hospital, Westchester Division; and joined the psychiatric staff of Cornell Medical School. His publications include papers on problems of catatonia, homosexuality, psychodynamics, psychiatric classification, hospital function, and the teaching of psychiatry. Dr. Sprague is a Diplomate of the American Board of Neurology and Psychiatry; and is a member of the American Psychiatric Association, Association for Research in Nervous and Mental Illness, and the American Psychopathological Association.

Probably, the most innate tendency of the living organism is to turn towards what seems pleasurable or satisfying and to turn away from whatever seems noxious or unpleasant. Kempf has written with illumination upon the mechanisms which he terms "acquisitive and avertive," through which the individual seeks to obtain as much as possible of gratification for the instinctive yearnings. Of these tendencies, positive and negative, it is intended here to discuss only the latter, the avertive pattern, the methods used by a person to escape from something which he believes or feels would be prejudicial to his best interests or his happiness. And even a further limitation of inquiry is planned. One may seek escape from unpleasantness by various means: there are the possibilities of attack, of flight, of rigid control, of toleration. One way of dealing with such problems is to deny them in whole or in part, and this mechanism alone will be dealt with here.

Denial, we find, has several shades of meaning. The dictionary gives such variations as these: rejection of a request; refusal to grant; refusal to admit the truth of, or assertion of the untruth of; negation; disowning. In studying this subject, we are plunged at once into the matter by asking such questions as: rejection of what request, made by whom, rejected by whom and to what end? What is not granted or indulged? If something be true, why seek to negate it? Wherein do such phenomena become pathological?

To answer such queries, it is necessary to review some of the facts of psychodynamics and to take into account the various elements which may enter into the situation. We may divide them first into the actual facts of the external world and the inner facts of the subject's personal psychology, and in turn, the psychological situation is a complex in which we may recognize the Id, the Ego, and the Super-ego. Also present within the individual are memory traces, some of which relate to actual past events, while others are of wishes, phantasies, hopes, etc. — things which never actually happened in the world of conduct, yet had a certain sort of mental reality and potentiality, and which had a value to the person.

At any given moment, there may be any of the following possible items toward which one will have to adopt some attitude, either acceptant or avertive, comfortable or uncomfortable: (1) physical facts of the present; (2) mental content now occurring as present fact, either conscious or unconscious; (3) memories which, though traces from the past, are themselves existing now; (4) instinctive urges or pressures in an ever-shifting combination, according to the varying degrees of insistence in each of the several components. All of these items, in fact, are constantly changing except for the memory traces, and even these change so far as their availability to consciousness is concerned.

In such a complicated intermingling of factors, there is one function which is always to be reckoned with. This is an Id function. It is the constant evaluation of the acceptability or inacceptability of the present status of the individual in terms of liking it or disliking it. Every possible occurrence is so sized up—not in a critical fashion, but in an unquestioning, naive, non-reasoning way, probably equivalent to the simple utterances "nice" or "bad." Such a decision is inherent in the organism where visceral sensations are concerned, but cannot be thought of as dealing directly and immediately with other issues without the collaboration and interaction of the Ego. That is, experience with the

outer world is necessary in order that a self can be established and enabled to sense its personal inclinations, to appreciate that "some one" feels this way. Also, it is only through such external experiences that the individual acquires knowledge of meaning—of the significance to be attached to phenomena—and of the ways available for so manipulating matters as to obtain the most pleasure and escape the most pain or displeasure. Thus it comes about that the Ego is kept busy dealing with the outer world, so that the Id will be maintained in as much comfort as possible.

About the time in the life development when the sense of self has become fairly well developed and has grown accustomed to the task of translating the raw formless longings of the Id into terms of specific real object relationships, there enters another complication. Instead of responding to some situation in such direct fashion as seems likely to give the desired Id gratification, one begins to consider other viewpoints which alter the earlier evaluations. Time was when one saw a pretty toy and innervated certain muscle groups in such a way that the object came into possession, causing thereby a pleasure which proved sufficient to validate and endorse that form of muscle innervation. But later the Ego has to learn about property rights and morals and self-abnegation, and finds itself saddled with a Super-ego which, in its turn, demands to be satisfied or to have its disquietude soothed.

From this time on, the Ego ceaselessly stands as the recipient of stresses demanding gratification from the Id and from the Super-ego and from the world of external objective reality demanding acceptance and compliance. This is no easy role to play, either from the standpoint of the numerous conflicts which incessantly arise, or from the viewpoint of the practically infinite complexity of facts, psychological and objective, which must be dealt with. We must be prepared to view this harrassed Ego from many angles: It represents the person as a synchronized entity, dealing with the world as an integrated unit. It stands as the agent and representative of the Id, fighting battles for its vague, formless urges in concrete terms. It serves as the medium of expression as well for the Super-ego dictates and longings. Likewise, it is the interpreter of the outside world, and because of its ability to manipulate, and so to discover meaningful relations, it is the function of the Ego to build up not only the immediate present contacts with reality, but also the symbolisms and indirect significances which characterize the mental life of an individual.

It would be simple enough if one had to deal with only one issue at a time, or if the values and meanings of a given phenomenon remained fixed, to react to it unequivocally and with a fixed responsive pattern. But this does not happen. It is not the single phenomenon to which we respond, but the phenomenon taken in relation to its setting, and to the meaning of its present concomitants to us. What under one group of conditions is acceptable, is not so with different accompaniments. The total situation alters the significances of the individual detail.

As long as such changes take place only in the external world, the individual's adaptation can be simply on the basis of meeting real facts as such. But whenever one or more of the factors involved is contained within the individual's own make-up there arises the need for an inner adjustment. All too often for comfort, there is some falling short of perfect contentment. At once conflict arises, and conflict is uncomfortable and discomfort awakens urges to alter the situation. Whether from the Id's desire for pleasure, or from the Super-ego's aspirations, or from the Ego's own wishes for its preservation undisturbed, these urges force the Ego to set about the task of bringing things to rights. This seems to be the setting and the pressure under which denial arises as one of the ways for striving to reduce the sum total of the distresses one is undergoing. Likewise the Ego, profiting from previous experiences, can appreciate the probabilities of the future towards which it may already start its reaction of facilitation or of antagonism in anticipation.

So it happens that we are given opportunities as physicians to understand, often in advance of any actual break in a person's mental health, what trends the person may sense as most probable and which as most inacceptable for him in the future. We find him developing certain patterns, personality

traits, speech mannerisms, choices of clothes, or marital partners, or occupations; and it is a fair inference that often such planning or selection may indicate the attempt at denying in advance the success of certain probabilities, just as the man who foresaw that he was likely to be attacked might practice with a punching bag and in so doing show us that he was striving to negate, i.e. to deny, the disagreeable likelihood of being beaten.

It has been hinted, just above, that one sometimes denies something in the future, as in saying: "If I get caught in a fire panic in the theater, I *won't* be a coward." Probably a great part of all our anxieties is met in this fashion. The Ego uses its knowledge and experience to make hypothetical pictures of coming events and situations. These are then studied, very often of course quite unconsciously, and what appears unsuitable is subject to corrective alteration. The unsuitability may be of two kinds: either the tentative picture is discordant with fact and truth, or it is not in harmony with one's conditionings for maintenance of happiness. In either instance, a tendency arises to retouch the picture, or to act in such a way as to avoid having it come out wrong. If it proves to be impossible to maintain factual truth and also to have affective satisfaction, there is the opportunity for the latter to be insisted upon at the expense of the former; if reality is sacrificed, gratification results, but we call the individual psychotic.

The issue with which the person deals may be a present, or a past circumstance—one which is presented by the immediacy of sensory experience, or one whose memory seeks to give it a present value. There is less flexibility in one's possible dealing with the present, of course, for the facts already exist. It is too late to contrive some way of molding them. If I foresee that Mr. X will be angry if I ask him certain questions, I may object to his wrath enough to refrain from asking and so the wrath will not occur. But if he confronts me this very moment and is now angry, I must somehow meet the discomfiting fact. And much less still can a past circumstance be manipulated. Where a regrettable memory recurs, the facts it recalls cannot now be made not to have happened. Will we be reality-minded, and philosophically accept the disagreeable past? Can we find in it another parcel of data for the Ego's store of experience, and take comfort in reflecting that we are now better sensitized and able to avoid similar mishaps in the future? If not, there is the possibility that our memory may make us so uneasy, so regretful, so ashamed, or give us such a distressing feeling of inadequacy that, unable to change the facts, we deny that these are the facts.

It could be said that the apparent facts are actually not facts. It might be claimed that the memory is mistaken, or that some additional material alters its significance. These ways of dealing with such a situation are used by all of us, but in mental illness, or shall we better say as the reason for our diagnosing a mental illness, there are to be observed more elaborate methods of solving the issues. Just as the longer range gun fires with a greater detonation, these more violent means of destroying displeasure have increased dangers and carry one further away from the average normal types of reaction.

Let us look at the actual content of some patients' reactions and gather some examples of denial patterns, in order to discover what principles they illustrate. From several case records at random the following material has been selected for our purpose: "I won't ever get any better." "I'm not crazy." "My bowels don't move; they don't ever move." "No, Doctor, I am not excited, not a bit of it!" "No one knows how I suffer." "Nobody was ever so wicked as I have been." "I'm dead; there is nobody here; I have no name." "There's nothing the matter with me." Patients refuse food, saying, "I can't eat." Others go into stuporous states, thus denying practically everything. "My clothes don't fit." "You don't understand my case." "You don't treat me right." "I'm not playing the man's part as I should." "You are not godly, pure and sincere." "I don't remember exactly and it troubles me." "I don't want homosexuality." "You're trying to make me confess things I never did, and I won't!" "I tell you, I didn't steal those clocks." "They aren't my parents; I don't know them; they're not interested in me; H. is not my name; I don't know whose it is, but it's not mine." "This is not the place for me; I can't be happy here." After telling

in detail his ideas about dictaphones in the walls, one patient stoutly denied he had ever thought of such a thing. One man referred to things he had said in a voice he could not hear, so he knew nothing about them. Patients escorted to the toilet refuse to release sphincters, only to relax them upon returning to bed. A patient who swore and struck consistently denied it even to those who had been attacked. Another spent months saying all day long: "It is not true. It is not. I am a perfect gentleman!" One would neither swallow nor expectorate his saliva, till it drooled by overflow. "You don't like me." "I couldn't pay for this whole hospital." And so on, ad infinitum.

By studying such trends in clinical records, a number of observations can be made. Denial occurs both in positive and in tacitly implied form. One patient will say, "I am not excited, Doctor," while another merely asserts that he is well now. But both are attempts of the Ego to avoid an uncomfortable realization of the failure of the longing for omnipotence which would overcome all weaknesses. He who insists, "I'm a quitter" is likewise making a denial of his capacity, and it seems that his Ego is being made to suffer by an exacting Super-ego. There are many examples of this Super-ego denial tendency, such as the pillar saints, the Hindu religious mortifiers of the flesh, the sacrificial mutilations and suicidal attempts.

Sometimes this simple pattern is complicated by some other mechanism, as in the following instances. A man who was horrified at the godlessness of his Ego drives toward homosexuality became so uncomfortable that he sought to ease his distress by accenting his own goodness and thus denying his "badness." Later he added a projection in which he denounced others for their absence of brotherly love (disguised homosexual attitudes) and denied that they were godly. His denial made it possible for him to voice his Super-ego criticisms without the pain of recognizing that the criticism was of himself. Also, it let him continue his deeply emotional interest in his fellowman, and for the very same reason as was originally the case, although now distorted by projection and reversal. Projection is thus a form of denial—one in which the formula reads, "this is not within me, but is in others."

We find that some of the items denied in mental illnesses are obvious truths, such as the fact that one has a head on his shoulders, while again the denial is of something so untrue as apparently not needing to be controverted, as with the plaint that one patient could not possibly pay for all the expenses of the entire hospital. Each of these cases seems to need explanation more than would some case more debatable, such as whether one did right to reach a certain conclusion. When the facts appear to be manifestly clearcut, why should tautology or absurdity seem useful to the patient? A clue is seen if we revert to the basic idea of all motivation, and say that it can be explained as an effort either to increase comfort and pleasure, or to decrease unpleasant or disquieting stimulation. It must be remembered that the actual wording of a patient's denial has two properties, that of meaning for its actual verbal content, and that of its structure or type value. One who persistently asserts that he is not a murderer may in his very words show us that he deals with a problem of sadism and its control. The man protesting his having to pay all the bills was not, however, in need of concern over finances. We should, therefore, be ready to study his denial as a structural pattern. It then can be discerned that in this way he seeks to avoid being held to account for something, that he feels somehow under an obligation which he wants to get free from.

This latter instance brings out another function of negation as well. It may frequently be employed as a means of getting Ego gratification by calling for repudiation from other persons. If they will contradict the patient's denial, it may be quite comforting. When a girl tells a man, "You don't love me any more," it may produce protestations, kisses and roses. When the depressed case denies that he will ever recover, it brings comfort to hear the physician's reassurances, as the restored case often confesses.

In the case of denials, which to the observer are patent misstatements, such as, "My name is not H," we may regularly expect to find some battle between parts of the individual's own psyche—some way in which some particular item has meanings or values

of conflicting nature within the personality structure. Thus, craving for autoerotism may spell gratification of sex instinct for the Id versus fear of insanity to the Ego. Denial of masturbation then could be a way of making more remote an issue certain to cause one or another kind of discontent whichever way it was settled. Denial that the bowels ever move is in some instances representative of the victory sought by the Super-ego in its struggle for cleanliness and purity against the childish pleasure of the Id in rectal evacuation. The monastic asceticism of some paranoid patients illustrates how in denying erotism the Super-ego vanquishes the lustful urgings of the Ego. On the other hand, a case of Ego overcoming the Super-ego's attempts at domination occurs in the amorous overactivity of a manic psychosis in a usually inhibited shy individual.

Inasmuch as we may very easily find examples of ascendency for each realm of the personality in its contests with each of the other personality realms, we are forced to the conclusion that the type of reaction in any given individual does not depend upon whether it be Id, or Ego, or Super-ego, which is involved, but rather—as in all battles—upon the relative strengths of the contestants. If then we observe a case with denial symptoms over some period of time, the variations in intensity of denial expression will give a rough indication of the change of balance in the inner struggle. When denial is no longer taking place, it tells us that no longer has it seemed necessary to use this method for coping with the inner problems because the one urge has gained, or the other has lost, some of its former vigor. The man who reiterates his protest, "It is not true; I am a perfect gentleman," finally experienced a sufficient subsidence of his homosexual instinctive craving that his Super-ego needed no longer to fear the outbreak of antisocial tendencies. Accordingly, it could lapse into a condition of contented security and did not have to seek the comfort of denying the hated and feared trend. Perhaps we do not give enough recognition to the fact of ever-shifting intensities in the Id's instinctive cravings, so that the balance between the various partial strivings constantly alters, and also the avertive and acquisitive patterns as well.

Some who are concerned over inacceptable instinct urges may be impressed, for some reason, by the bodily aspects of the situation. These will naturally direct their disavowal towards the body and the physiological functions. Such persons protest the possibility of normal physiological activity. They cannot eat; the elimination is at fault; the stomach is gone, or is solid. Or they refuse themselves the use of their functions—a kind of conduct denial instead of the spoken or thought form. Here are to be seen those who are mute, stuporous, deliberately withholding of excreta, negativistic, automatically obedient. The persons who consider the psychological aspect of their problems more are apt to express denial in those terms: this is not their own mind, or personality; the thought content is not theirs but is foisted upon them; they have no privacy of thought; control of ideas has gone; they are no longer sane; their minds are gone; they are not responsible for their conduct; they do not even exist any more, but this is someone else, etc.

Denial is not infrequently employed to lessen one of the very oldest and most painful of traumata. From an early age the Ego is frequently being distressed by fresh evidence that it is not so powerful as it had supposed and wished. The omnipotence desire is such a firmly entrenched one in the Ego that it is never abandoned any further than facts demand. Any item in one's mental illness which threatens to imperil it further is liable to denial if the discomfiture should prove too severe. "Saving one's face" is a familiar expression for this method of seeking to deny whatever offers to do further damage to one of our most dearly beloved myths. Accordingly, our loud impulsive manic assures us he is not ill, not impulsive. The confused schizophrenic asserts that he is not crazy, or even that this person is not actually himself. The would-be sturdy, self-dependant man crippled by poliomyelitis denies his limitation by assumption of dictatorial powers and high-handed rage patterns. One man laments that he is not playing a real man's part in life. The husband of a famous woman denies his own lesser position by making fantastic demands and assailing such unfair treatment as not giving him his way about things.

Disavowal, contradiction, or denial, then, means discontent over some issues. It means that the individual has felt constrained to go on the defensive. As this is an unpleasurable situation, it leads to efforts at removing the need for feeling discomfitted. The wish that matters were not as they are leads to the assertion that they are not so—a simple accomplishment of the omnipotence of thought which we carry traces of throughout life. If there is a gain derived in such a procedure, it is at the price of a loss in reality appreciation. The implications derivable from these brief formulations give suggestions as to therapeutic approaches, but those issues must await discussion on another occasion. In conclusion, one may point out that the physician seeking clues to his patients' problems of conflict, defense and denial will do well to consider a well-known phrase: "Methinks, my lord, he doth protest too much."

121 Westchester Avenue.

Treatment of the Nervous Patient*

ROSCOE W. CAVELL, M.D.
Eloise, Michigan

Dr. Cavell received his M.D. degree from the College of Medical Evangelists in 1921. Since 1936, he has been an Associate Psychiatrist at the Eloise Hospital. Dr. Cavell is a member of the American Psychiatric Association and the Michigan Society of Neurology and Psychiatry.

Every other patient that comes to the office of the private practitioner does so because of a disorder that is functional in origin. I am not the authority for this statement, although my own experience in general practice makes me feel that it is a conservative estimate. Many men, prominent in the various special fields of medicine, such as Hugh Cabot and Foster Kennedy, have made similar estimates. The medical student meets the neurotic the first day he enters the out-patient department and is confronted by the same problem on the last day of his practice. Dr. Walter Alvarez tells this story on himself. When he first went to the Mayo Clinic and began work in the out-patient department, he noted under the space for diagnosis the letters "G D N." He puzzled over this for some time and finally asked one of the other men in the Clinic who replied, "Oh, that means God damn neurotic." This attitude toward these unfortunates has changed little in recent years, although our knowledge of the dynamics of the psychoneuroses is much greater than it was ten or fifteen years ago. I am going to attempt to bring to you a few points that I hope will be helpful to you in treating these "neurotics."

The term "neurotic" is applied not only to patients whose disability is functional or psychogenic in origin, but to many others who have organic diseases, but who react to them in a psychoneurotic manner. I wish to emphasize right here that because a patient has been labeled neurotic is no assurance that he is not suffering from an organic illness, either acute or chronic, as well. I was somewhat embarrassed very recently by a patient of this type. She is a 43-year old woman who has a post-encephalitic syndrome. She makes the usual neurotic complaints and always desires an examination of some sort. One day her throat will be sore; then a pain in the chest; and then various abdominal pains. She has had a very thorough and complete examination on several occasions, including cystoscopic examination, a G. I. series, x-rays of the chest, etc. I saw her on my ward rounds recently and she made the usual complaints, this time of abdominal pain and also of urinary frequency. This was nothing out of the ordinary and the nurse stated that she had not seen the patient in the lavatory any oftener than usual. I took it for granted that it was her neurotic behavior and ordered a placebo. The next day, however, the patient had a temperature of 102°, and the urine was loaded with pus. Adequate, and fortunately, satisfactory treatment was instituted and the condition rapidly subsided. It has been several weeks since this occurrence, but the patient still makes frequent complaints of pain, burning, etc., although the urine has been entirely

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free of pus for some time. She now includes these symptoms, by which she gained considerable attention, with her other psychoneurotic ideas, although the original organic cause is no longer present.

I have not defined the term "neurotic" because I am sure you are all familiar with what is meant by it. I dare say that the mention of the word brings a mental picture of from one to a dozen patients that are under your care at the present time and that you probably characterize, mentally at least if not out loud, as "pains in the neck." I am reminded in this connection of the story of young Dr. Jones, who after completion of his internship and a three-year residence, joined his dear old dad in a little office out in the sticks. Dad was easily persuaded to take a well-earned and much needed rest, but on his return immediately inquired about the welfare of his patients. The usual fractures and O. B.'s were mentioned and then young Dr. Jones stated, with a great deal of pride, "You know, Dad, I spent a lot of time on that confounded pest, Mrs. X, but I have cured her neurosis." After a moment's thoughtful consideration Dad replied, "Well, son, I am very proud of your success and Mrs. X was certainly very deserving of your very best efforts. Her neurosis paid your way through medical school."

The problems of pathology, etiology, and treatment confront us in the neurotic patient just as they do in the patient suffering from an organic illness. The answers to these questions, however, are not as easy in the neurotic. The cause of the neurosis is often very obscure and the treatment is difficult, and seldom fully satisfactory. I use the term "neurotic" in labeling this group of patients because I am sure it brings to the minds of all of you about the same picture. Psychoneurosis is, of course, a much more scientific classification and is the term that we all use when we are on our dignity, neurotic being just a sort of nickname that we can use for informal discussion. I am sure you are not interested in the splitting of hairs used in the sub-classifications of the psychoneuroses and neither are you interested in three and four syllable words, so for the remainder of this paper, at the risk of being considered unscientific, I am going to use the term "neurotic."

Physicians and laymen alike are very prone to feel that the neurotics themselves are in a large degree to blame for their illnesses and that they can prevent them if they exercise a little will power. They are also frequently accused of malingering. These attitudes are most unfortunate, as the patient is a real sufferer and is no more a malingerer than the patient with pneumonia or any organic disease. The psychoneuroses (neurotics) comprise a group of relatively benign mental disorders in which the symptoms are often expressed as physical manifestations, but are due entirely to mental forces, and are intelligible only in psychobiological terms. The symptoms presented by the neurotic solve some particular problem for him and, therefore, serve a useful purpose. It is one of the mechanisms by which the organism adapts itself to its environment just as the right rectus rigidity in appendicitis is nature's way of splinting the inflamed appendix. The resulting behavior may not be socially acceptable, but the mental splint protects the personality against sudden harm whether that harm is coming from the environment or from some inner mental conflict. The symptoms seen in the neurotic, therefore, must be considered as the reaction pattern with which this individual meets his life's problems. Neurotic behavior is just as important to the integrity and safety of the organism as is the behavior of the leukocytes when they combat an invading micro-organism. The walling off of this micro-organism with the resulting abscess formation and slough followed by scar tissue, may damage the future usefulness of some portion of the organism, just as the neurotic behavior pattern may damage some part of the personality, but in both cases the organism as a whole is protected from threatening danger. These points are stressed because it is necessary to keep them always in mind when treating neurotics.

We have no specific therapy for the cure of these unfortunates, and nature was very unkind to the physician when she did not design them as self-limiting diseases. It is very difficult to outline a concrete and practical plan of therapy. The diagnosis is not usually very difficult, the important thing being of course not to overlook the presence

of definite organic disease. This disease, if present, may be the thing to which the individual is reacting in a neurotic manner or may be purely a coincidence. The neurotic, of course, is subject to all of the organic diseases that anyone else is subject to, and yet the presence of an organic illness may play no part at all either as a cause or in the course of the neurosis. The disorder results from the environment. Treatment must therefore be directed toward changing either the environment or the behavior. It is usually very difficult and many times utterly impossible to change the environment. One cannot make an "Oomph" girl of the unfortunate female whose face and figure only a mother could love. Neither can we supply the intelligence which nature failed to give the young man whose ambition is to be president of the firm, but whose mental caliber keeps him in the laboring group. Our only alternative, then, is to try to build up in these individuals a more healthy attitude toward that environment which we cannot change. All neuroses, of course, do not arise from the inability of the individual to adjust to his environment. The mental conflict coming from within the individual because of the instinctive urges or drives and the inability to gratify these is the major cause of most of the neuroses. The combination of all of these factors are present as causative agents in most of the neurotics that we see.

Treatment of the neurotic, therefore, must be along psychobiological and psychotherapeutic lines. But what are the specific psychotherapeutic and psychobiologic measures that we can use? My own experience during a number of years in general practice in trying to find the answer to that question is probably the thing that led me to attempt to give a paper of this kind. I do not have the answer to the question, but I feel that I can give you a few ideas which may be helpful.

How does one proceed once the diagnosis has definitely established that the individual patient is neurotic. The first prerequisite is the realization that the treatment is going to be long continued and cannot safely be shortened. The physician must also acquire a healthy attitude toward both the neurotic patient and neuroses in general. I mean by this that we must not dislike the neurotic because of our inability to promptly cure the condition of which he complains. The physician must not project his own frustrations upon the patient; that is, being confronted with something that he cannot successfully treat he must not blame his lack of success on the patient, feeling that it is the patient's perverseness and not his own lack of ability which has prevented the cure. The time factor is important in all psychopathological disorders for as a rule they are much more extended than organic disease and, as I stated before, they are not self-limiting. A new definition of the word "treatment," or rather a different definition, may have to be formulated. Webster's Dictionary defines treatment as "the management and care of a patient for the combatting of his disorder." The reeducation of the patient in order to help him accept the undesirable things in his environment falls well within the limits of this definition. Attempts to change the environment are, of course, accepted methods of treatment in many of the organic diseases.

I am going to attempt to outline a very broad and very general method of procedure in the treatment of the neurotic. The patient comes to you complaining of pain or other physical disability. The examination has revealed no organic cause for this condition. How then can we proceed in the treatment of this patient. First, *the patient must not be told, "there is nothing wrong with you."* They have undoubtedly been made to feel, both by the family and other physicians, that they are in some way to blame for their condition. It is equally useless then to say, "Now this trouble you have is not at all serious, but if you don't buck up and lick this thing, it will be serious." This approach only makes the patient's unconscious more determined to make the complaint impress the world as a serious one.

Most patients will not accept, at least early in the treatment, the fact that their condition is not an organic one. Getting them to accept this fact is one of the most difficult problems in the entire treatment. The physician must first listen to the patient's complaints. This sounds trite, of course, but in the treatment of the neurotic patient it is most important, for the very telling of their troubles to a sympathetic listener is a definite

relief in itself. After a very little experience, the physician can soon learn to pick out the important points that he will wish to stress later on in the case. The existence of the complaint must then be justified. Again, let me emphasize that we must not tell the patient that the trouble is just "nervousness" or that there is no organic cause for it. We must enlarge upon the complaint, as the patient gave it, sufficiently to include its psychic aspect. As an example, I am going to give a dialogue which may be helpful. The patient has complained of gastro-intestinal discomfort, nausea, vomiting, etc. The physician is attempting to tell this patient that the condition is not an organic one. "Now, Mrs. X, you have told me that your complaints interfere with your work and cause you a lot of worry and anxiety. You have told people, of course, not to worry. You have told many of your friends that worry is bad for them. You know, therefore, that your mental attitude interferes with the normal work of your stomach. Now, our problem is to find out how much of your stomach difficulty comes from the stomach itself and how much your stomach is interfered with by your usual activities. You expect, of course, an upset stomach when you are worried. Now, we will have to find out first how much of your stomach is handicapped in its work by your worry, fears and anxieties. It may be that if you had no worries or anxieties, your stomach would be able to carry on its work without any difficulty." We can thus establish a definite justification for the patient's complaints but at the same time we have led the patient to accept the fact that the difficulty is not organic but psychogenic.

The history should be orderly and systematic, and the personality problems as they appear in this history should be stressed so that the patient may be desensitized in order that these traits may be investigated further at a later date. It must always be kept in mind that the neurosis serves a purpose and that the patient's unconscious is ever on the defensive against any threats to its security. There may be many psychological and other varieties of "skeletons" in the closet that have not come to light in the examination. If these are dragged out at this time, the patient may become very skeptical and be discouraged at the outset and even antagonistic to the physician. The psychic factors involved in producing the disorder can usually be brought out in later interviews by pointing out in a similar way how they effected the patient's complaints.

The matter of prognosis and its implications is a decidedly difficult one. The responsible relatives of the patient should, of course, be given the facts as they stand. Reassurance for the patient should be unstinted if he is in the throes of a major reaction, especially a depression or state of anxiety. In general, however, with the exception of acute anxiety states and those neurotics who show considerable positive suggestibility, it probably is well to make the patient feel his responsibility for getting well. One, therefore, must be cautious about giving too much reassurance in order to prevent the patient from being content to just ride along, leaving all the initiative to the physician. The financial obligations may be used in some cases to increase the patient's willingness for constructive cooperation. The role of the time factors in both prognosis and treatment should be carefully explained. If the therapeutic period is rigidly fixed, the patient begins to anticipate results and becomes dissatisfied with his evaluation of the progress, getting more tense, thus bringing in a new complication. The preliminary formulation should also include a word of explanation about possible variations both in the manifestations of the illness and in the possible methods of treatment.

The prerequisite, so to speak, of the treatment being now fulfilled, we can proceed to more definite therapeutic measures. For this purpose we can divide the therapies into two forms, the direct and the indirect. The indirect therapies are those used in correcting the varying manifestations of the disorder. They may be divided into symptomatic or palliative measures, supportive measures, and those which help the patient to sublimate. Under palliative or symptomatic measures are included those which help to relieve pain, insomnia and, of course, the correction of any existing organic condition. The supportive measures include those that are necessary for the improvement of the patient's general physical condition, such as the relieving of

focal infections, anemias, dietary deficiencies, etc. The sublimative therapies include special attention to occupational and recreational activities, or any measures that help to bring about a better balance in the patient's living condition.

The direct therapies include the management of the factors in the external environment. The patient's output of energy must be balanced in terms of his individual capacity as well as in the terms of the expectations of what others think he can do. Unhealthy attitudes, misconceptions and ideologies of family, employer, school, community, etc., must be modified insofar as it is possible. These procedures are not curative, but they do help to relieve and remove some of the dynamic factors involved in the production of the illness. The management of the personality determining factors includes the straightening out of various memories and of the patient's ideas and misunderstandings which have been problems in the patient's utilization of his assets and capacities. We must help to correct anything that gives rise to attitudes and reactions that interfere with the satisfactory, healthy and efficient functioning of the organism. This treatment varies with the personality of the patient, the type of illness from which he suffers and the physician's own personality and training, and such impersonal factors as time, money, etc. We can further divide the management of these personality determining factors under three headings; first, suggestive measures; second, personality study; and third, re-education.

The suggestive therapy is always used to some extent in the form of reassurance, persuasion and indirect and direct suggestion. These measures are all utilized from the time the patient first confides the story of his complaints to the physician, clear through the completion of the examination, formulation of the problem and the treatment period. Direct suggestion may be of some value in a few of the minor psychoneurotic reactions. Personality study consists of analysis of the personality attributes. These may be outlined as follows:

1. General personality survey (jobs held and attitudes toward them, hobbies, friends, religion, education as examples of performance).

2. Special survey of the psychobiologic assets (intelligence and memory functions, emotional rises and balance).

3. Range and fluctuation of fitness with regard to work, play, rest and sleep.

4. Social relations within the family and relative role of self and of social dependence.

5. Sex development.

6. Difficulties and handicaps.

7. Specific disappointments and reactions to them.

8. An enumeration, in the form of an index of the events and situations in life which constitute special problems or determining tendencies, as a result of the personality study.

This is carried out by means of confidential interviews in which the physician takes the lead and directs the discussion. This does not mean that the doctor does the talking. On the contrary, the exact opposite is most desirable, as the doctor should always be the sympathetic and understanding listener. These patients are always willing and ready to keep their appointments once they have been made to feel that they have found a source of help and that they are not being blamed because they are ill. Fifteen to thirty minute interviews, if the physician just listens understandingly, will effect remarkable, and often permanent, improvement. These interviews should be held when the physician is least apt to be interrupted and should be charged for the same as any other call in which the same amount of time is used.

The patient's symptoms are considered and evaluated, the problems recognized as significant by the physician are talked over and the patient's memory, his imaginations and anticipations, his urgings and strivings, his attitude toward these drives as well as toward the past, present and future, are thoroughly discussed. The patient is thus led through a systematic and controlled process of ventilation, and we hope, becomes affectively desensitized to certain topics and factors. At the same time, the patient is led to find ways and means of developing modifications for his use of past experiences and of attitudes pertaining to his present and of ambitions and anticipations of his future, and a more efficient self-management of his present situation. The concluding period of

each interview should be used to evaluate the patient's progress in terms of the original complaint. The goal of each interview should be that of coordinating the various factors of the personality and of the external environment into something that offers the patient security and concrete facts with which to guide and control his future performance. It is important that the patient should not be overwhelmed with too much or too difficult material at one time. Appropriate rest periods may be allowed when indicated and the physician must feel free to alter his planned therapy to meet any difficulties that may arise. If the patient cannot or will not cooperate because of his personal rigidity or resistance to the procedure, this fact must be brought to his attention and discussed in the hope of modifying the difficulty. If the symptomatic and supportive measures have ameliorated the symptoms to the extent that the patient loses interest or incentive to continue his treatments, it is often necessary to change these therapeutic measures or make less use of them until the patient has reoriented himself to his actual status by allowing the symptoms to again come to the fore. The object of the treatment, of course, is to return the patient to his usual, or rather a more useful and less invalid condition. The physician must, therefore, not allow himself or permit himself to continue the treatment too long. It is not necessary for the patient to understand all of the unconscious mechanisms involved in his illness, the physician's objective being merely to find or rather to help the patient find a more acceptable and less harmful way of meeting either the environmental or psychological conflict.

In conclusion, I wish to re-emphasize one or two points. First, the length of time necessary for the treatment cannot be stated definitely. You can tell the patient with a Colles fracture that you will take off the splint in four weeks and he can return to work in six weeks, but no such definite time limits can be given the neurotic. Second, the final results are seldom completely satisfactory. The method I have outlined, however, provides a rational method of approach, by dealing with the causative factors so far as they are known. If carried out with understanding and patience (no pun intended), you will relieve much human suffering and rescue from the clutches of the charlatan many a grateful and profitable patient.

Eloise Hospital.

Treatment of Paralysis Agitans *(Parkinsonism)*

HENRY A. DAVIDSON, M.D.
Newark, New Jersey

Dr. Davidson received his M.D. degree from the Jefferson Medical College in 1928, and his M.S. in Neurology from the University of Pennsylvania in 1931. He is Adjunct Neurologist at the Beth Israel Hospital in Newark, and Assistant Neurologist at the Newark City Hospital. He is a member of the Association for Research in Nervous and Mental Disease, and of the American Psychiatric Association, as well as Secretary of the New Jersey Neuro-Psychiatric Association. Dr. Davidson has, for the last six years, contributed the article on "Encephalitis" to the "Review of Medical Progress," and has published more than a dozen original articles in the field of neuropsychiatry.

Paralysis agitans (parkinsonism) is a common disorder. Relatively few of its victims show the "pill-rolling" tremor of classical description. What the practitioner usually sees is a slowed-down, rigid, tense patient, with immobile face, stiff posture, wooden gait, lost or markedly diminished associated movements, and a monotonous voice punctuated by explosive stammering.

Four effective drugs are available: hyoscine, stramonium, benzedrine and atropine. The attached table schematizes the therapy. It is necessary to plan long-term treatment, and during the first year the physician should experimentally vary the drugs. By the end of this year, the patient himself will tell the doctor what medication he prefers. And the patient, too, will elect whether to tolerate the untoward effects of increasing dosage or whether to free himself of these complications at the expense of diminished therapeutic benefits.

Hyoscine (Scopolamine)

Hyoscine (or Scopolamine) is usually given as the hydrobromide. In my experience, at least, most patients ask for hyoscine when, after contact with all four drugs, they are queried as to which they prefer. A good starting dose is gr. 1/150 twice a day. This may be increased or decreased according to the patient's reaction. Blurred vision demands a reduced dosage, though not infrequently patients insist on the larger amount in spite of the complication. Patients with tremors and oculo-gyric crises are especially helped by hyoscine. For post-encephalitic cases, some of the other medications may be found more effective. Arteriosclerotic patients, however, generally report better results with hyoscine.*

Atropine

Atropine is always worth trying. When it helps at all, it gives the patient a feeling of well-being not attainable with any other drug. It is especially useful when salivation and rigidity are the bitter complaints. It may be administered in tablet or solution form. Tablets should contain 0.25 mg. (gr. 1/250) of atropine sulfate. If a water solution is used (and this is generally preferred because it permits a more flexible dosage schedule), it should be in 1/200 (that is about 0.5%) strength. One drop of this solution delivers about 0.25 mg. of atropine and is therefore the approximate equivalent of a gr. 1/250 tablet.

If tablets are used, start with one a day, and increase gradually until the patient is taking as much as he can tolerate. Hall[1] has given 15 or even 18 mg. a day. (Of course, if this amount is reached, it will be more practical to have 2.0 or 3.0 mg. tablets prepared.)

Most patients prefer the solution to the tablet. Initial dose is one drop three times a day, increased a drop a day until the patient is taking 45 or 50 drops a day—unless intolerable dryness of mouth or blurring of vision necessitates a lower maximum. Much has been said about the "decoction in white wine" made from Italian or Bulgarian Atropa root, instead of the water solution made from the standard Atropa.* However, the white wine is difficult to prepare and the foreign preparations are usually available only in larger cities. I have not found the Bulgarian or Italian preparation or the white wine decoction any more effective than the ordinary 1/200 water solution of the standard belladonna alkaloid.

Tincture of belladonna is not effective.

Atropine may be given hypodermically, starting with gr. 1/300 increasing gradually to gr. 1/150, once a day. Since treatment must be continuous, it is inadvisable to use this parenteral route unless the oral medication provokes vomiting.

Benzedrine Sulphate

Benzedrine (Amphetamine) Sulphate is especially helpful in patients whose chief complaint is fatigue, drowsiness, or weakness. The ten-milligramme tablet is used, with an initial dose of one tablet a day, and a gradual increase of half a tablet every 3 or 4 days. Solomon and his colleagues[2] have given as much as 160 mg. (16 tablets) a day. However, I find that most patients complain of increased tension and cardiac palpitations long before this dosage is reached. The drug is usually ineffective in old or arteriosclerotic patients; but it is always worth a trial in post-encephalitic or toxic cases of parkinsonism. Solomon's group[2] found that it effected a disappearance of oculogyric crises in 75 per cent of their patients. While my own experience has not been that encouraging, I have found that it usually reduced the frequency of these crises. Combined with stramonium, benzedrine is an especially good medication, since the former seems to relieve the rigidity while the benzedrine gives a feeling of energy and general well-being. Benzedrine should not be taken in the late afternoon or evening as it may cause sleeplessness.

Stramonium

While Stramonium is botanically similar to belladonna and hyoscyamus, it differs therapeutically in that the whole drug and

* Hyoscine should not be confused with hyoscyamine. Both are alkaloids of hyoscyamus, but the latter is an antispasmodic not especially useful in parkinsonism. Hyoscine is the same as scopolamine.

* The standard preparation is either of Western European origin, or comes from the cultivated American Atropa plant.

not just the alkaloid is used. Stramonium often dramatically relieves the feeling of stiffness. Either the pill or tincture may be used, one minim of the latter being pharmacologically equivalent to a grain of the leaf or pill. Although text-books suggest that the average dose of tincture of stramonium is 10 to 15 minims, these amounts are useless in paralysis agitans. It is necessary to start with a daily total dose of about one dram (20 minims td) and to increase it gradually—by 5 or 10 minims a week. Dosage must be pushed to tolerance before expecting results. While I never dared exceed 3 drams a day, Hodemaker and Burns[3] found an average dose of 75 minims *three times a day* tolerable and effective. The excessive dryness which results can be ameliorated by the daily administration of gr. 1/10 of one of the pilocarpine salts. The blurred vision provoked by the mydriatic effect of the stramonium, however, is not controllable except by reducing the dose. The choice at this point may be left to the patient. As often as not he prefers the blurred vision, due to the medication, to the rigidity which returns when dosage is diminished. As suggested above, stramonium may be combined with benzedrine. If benzedrine is not used, it is good practice to combine the stramonium with the daily administration of gr. 3/8 of ephedrine sulphate, especially in post-encephalitic cases.

A 2½ grain pill of stramonium leaves is also available. Best routine is to start with one or two a day and increase gradually to 3, 4, or even 5 pills a day, if the patient can tolerate it.

Precautions

It is not good practice to give sedatives in addition to the specific medication. If supplementary sedation is needed, try calcium lactate or, if this is ineffective, administer bromides. Do not give barbiturates (phenobarbital, barbital, or any of its compounds) as this may aggravate the rigidity.[4] Avoid benzedrine in patients with hypertension, as well as in patients complaining of insomnia, as the drug tends to raise blood pressure and to interfere with sleep.

Managing the Patient

The actual management of the parkinsonian patient is a three-fold problem. Elements in the management job are: (1) psychotherapy, (2) physiotherapy and (3) the drug routine.

Psychotherapy: Victims of paralysis agitans often try the patience of the practitioner. They bombard the doctor with the same questions over and over again. They demand more prompt relief, and complain bitterly if improvement is not spectacular. Thwarted by their own slowness and stiffness, they may become hostile or unpleasantly aggressive on the one hand, or depressed and self-pitying on the other. It is therefore necessary for the physician to maintain an optimistic calm in dealing with these patients. The doctor should pounce on every evidence of improvement, no matter how slight, and present it triumphantly to the patient. "You see how your eye-turnings have become less frequent!" or "that hand is certainly less shaky than it was last month!" It is necessary constantly to reassure the patient that the symptoms will be controlled. And it is important to pry the patient away from any feeling of invalidism. "Will I ever be cured, doctor?" they repeat. The question cannot be answered honestly without provoking a good deal of bitterness and depression. Better therefore to evade with: "Well, I don't know that you'll ever be as spry as you were when you were younger; but I certainly hope to have the shaking, the stiffness and the eye-movements under control." The disease, remember, is an emotional as well as a physical experience. The doctor must make some effort to evaluate what it means to the patient emotionally, and to help in this as well in the more mechanical aspects of the disorder.

Physiotherapy: The patient must be encouraged to "limber up" the stiffened extremities, to swing his arms deliberately as he walks, to use his joints freely, swinging them through as wide an arc as practical, and to do these things purposefully and consciously until they become habitual. He must actually retrain his movements to allow for the stiffness and tremors, and he should be encouraged to go systematically about this re-educating of motion-patterns. Warm tub baths leave a feeling of relaxation for a few hours, and the patient should be told to bathe

daily, and to take additional warm baths prior to any occasion when he wants to feel his best. Massage is helpful in teaching the patients how to "limber up" their musculature.

Drug Routine

Each practitioner will work out the drug routine most effective for his patient. It is good practice to try all four of the drugs in the course of a year, using one for 2 or 3 months, then another, until all four have been tried. A patient of average intelligence can be asked to keep a sort of medical diary, noting each week the apparent effect of the medication on the chief symptoms. The number of oculgyric crises experienced during the week should be recorded or tabulated. To discourage excessive introspection, the patient should be asked to keep weekly not daily notes. At each visit, the physician can enter a summary of the patient's notes in the medical chart, and thus build up a rich workable experience on the effect of the several medications. My observation is that regardless of the dosage prescribed, patients will make adjustments in the amount of medication according to their own experience anyway, and for this reason the subject's own notes should indicate how much medication he actually took, rather than the amount suggested by the doctor.

Which preparations should be used first? The following suggestions are given not as a rigid blueprint of therapy, but merely as a set of practical hints to the practitioner bewildered by the conflicting claims of advocates of the several drugs:

Arteriosclerotic and Senile Cases: Hysocine is the best starting drug, if tremors or oculogyric crises are the conspicuous symptoms; stramonium, if slowness and stiffness are the bitterest complaints.

Post-Encephalitic and Toxic Cases: Benzedrine is worth a trial as the initial drug, especially when drowsiness or weakness are the prime presenting symptoms.

Oculogyric crises: In all but arteriosclerotic cases, benzedrine is the drug of choice for reducing the frequency of oculogyric crises. In arteriosclerotic patients, hyoscine should be tried first.

Salivation and Drooling are controlled more promptly by atropine than by any of the other drugs.

Tremors: Hyoscine first, and atropine later,

DRUGS USED EFFECTIVELY IN TREATMENT OF PARALYSIS AGITANS

(See text for details of dosage routine)

DRUG	HOW PRESCRIBED	DOSAGE	CHIEF EFFECTS ON
ATROPINE SULFATE SOLUTION	1/200 solution of atropine sulfate in water. One drop=gr. 1/250.	One drop t.i.d. Increase very slowly to 10 or 15 drops t.i.d.	Salivation. Malaise. Tremors.
ATROPINE SULFATE TABLETS	1 tablet=gr. 1/250. =¼ milligramme.	One tablet t.i.d. Increase very slowly to 10 tablets t.i.d.	Salivation. Malaise. Tremors.
BENZEDRINE SULFATE TABLETS	1 tablet=10 milligrammes.	One tablet a day, gradual increase to 10 or 12 tablets a day.	Drowsiness. Weakness. Oculogyric Crises.
HYOSCINE OR SCOPOLAMINE HYDROBROMIDE	Tablets of gr. 1/50 gr. 1/100 and gr. 1/150.	From gr. 1/150 to gr. 1/50 a day, according to response, starting with smaller dose.	Tremors. Stiffness. Oculogyric Crises.
PILLS OR TABLETS OF STRAMONIUM	Pill=2½ grains of stramonium leaves or 25 minims of the tincture.	One to four pills a day.	Rigidity. Stiffness.
TINCTURE OF STRAMONIUM	One minim=1 grain of stramonium leaves.	20 minims t.i.d. with slow increase to about a drachm three times a day.	Rigidity. Stiffness.

constitute a good routine for excessively tremulous patients.

Stiffness and Slowness respond best to stramonium, which should, therefore, be the first therapy when these symptoms are the chief complaints.

Weakness and Drowsiness are most effectively controlled by benzedrine, except in the presence of definite hypertension.

Combination of drugs: While I have tried giving three medications simultaneously, this is, I think, bad practice. It overloads the patient with potentially toxic drugs and confuses the doctor when he is trying to sort out the effects of each medication. Two drugs at a time should be the maximum pharmaceutical burden imposed on the patient.

Order of Administration: After 2 or 3 months of one drug or combination of drugs, the doctor should make a sudden change, carefully noting the effects of this shift. It is not wise to evaluate results of the change until the patient has had several weeks experience with the new combination. During the first six months of treatment, it is more instructive to change both drugs (if two are being given) than to replace only one. After all four have been tried, the doctor can, by reviewing his own notes and the patient's, formulate a more or less permanent treatment regime. Of course, the appearance of untoward toxic symptoms is a signal for a lowering dosage. If this reduction wipes out the therapeutic effect, it will be necessary to change to one of the other drugs.

31 Lincoln Park.

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Further Observations on Psychoses in Relation to Head Injuries and their Classification

EUGENE DAVIDOFF, M.D. and PAUL HOCH, M.D.
Syracuse, New York *New York, New York*

Dr. Davidoff was born in 1901 and graduated from the Iowa University College of Medicine in 1927. At the present time, he is Assistant Professor of Psychiatry at the Syracuse University College of Medicine and the Senior Clinical Psychiatrist of the Syracuse Psychopathic Hospital. He is affiliated with four of the outstanding medical societies of the country, among them the American Psychiatric Association and a branch of the American Board of Psychiatry. His writing include studies on amphetamine sulfate, traumatic and alcoholic psychoses, psychoneuroses and somatic disease and juvenile delinquency.

Dr. Hoch was born in Budapest. He graduated from the University of Goettingen in 1926. Following his internship and residency at the University of Goettingen, he became Assistant at the Psychiatric Clinic at Zurich-Burgholzli, and later First Assistant and Privatdozent for neurology and psychiatry at the Neuropsychiatric Clinic, Goettingen. Since 1933, he has been on the staff of Manhattan State Hospital and the Hospital for Joint Diseases. In the latter, he has been in charge of the Mental Hygiene Clinic. Dr. Hoch is a member of the American Psychiatric Association, the American Psychopathological Association, and the New York Society of Clinical Psychiatry. He has published numerous articles in the field of neurology and psychiatry.

In an earlier paper, read in 1935 and published in condensed form recently, two factors were considered by us as important in determining the severity of post-traumatic mental manifestations: (1) the extent and nature of the injury and the subsequent brain lesion; and (2) the integration of the previous personality of the individual.

In our unpublished data we observed that the brain injury, if severe, may be the paramount etiologic factor. However, the trauma may be only part of the etiologic constellation. Moreover, in other instances despite the history of head injury, careful study may show that the trauma plays a minor or negligable role in the causation of symptoms. This is particularly so when the injury is a mild one and there is a pre-existing psychosis.

Therefore, we further classified the mental states caused by head trauma as follows:

(1) The acute stage, including confused and delerious states.

(2) The subacute or Korsakow's type.

(3) The chronic states.

A. Post-traumatic mental deterioration or defect where organic factors predominate and where the previous personality plays a relatively lesser role in the symptomatology.

B. Post-traumatic personality disorder type where the previous personality is a relatively greater factor in determining the course.

C. The combined type in which factors (A) and (B) are of equal importance.

D. Traumatic neuroses.

(4) The mixed type which is complicated by (A) a co-existing, pre-existing, or subsequent organic state which clouds the picture, or (B) a pre-existing, co-existing or subsequent functional psychosis, psychoneurosis or psychopathic state.

(1 & 2) Acute and subacute stages (post-concussion syndrome)

Nearly all traumatic psychoses presented in the acute early stage display either a period of unconsciousness and confusion or a profound delirium with marked clouding of consciousness, associated with restlessness or stupor, incoherence, and psychosensory and delusional disorders. Many of these cases may recover comparatively rapidly after the acute stage subsides. On the other hand, they may display, soon after, symptoms indicative of the subacute Korsakow's type with fabrications or more prolonged confusion. Later, they may emerge into the chronic state of post-traumatic mental deterioration or post-traumatic personality disorder. Early evaluations of the acute phenomena may be misleading and unreliable. The prolonged Korsakow's syndrome is merely the stage which, in many instances, is a connecting link between the confusion or delirium, and post-traumatic deterioration.

(3) Chronic States

A. *Post-traumatic mental deterioration:* Because of the presence of the organic defect syndrome which included intellectual deterioration, emotional instability, neurologic and encephalographic findings, the recognition of this sub-group is not too difficult. This is particularly true if an adequate history is obtained and the chronic manifestations emerge following the acute and subacute manifestations noted above.

A post-traumatic regression may occur in an apparently well adapted individual who reacts poorly to the traumatic situation because of the organic changes occasioned by the head injury. Those who are engaged in skilled occupations or who work under tension, those who have been suffering from somatic disease, those with specific abilities or disabilities, and those who have had previous head injury may adjust poorly following the trauma.

In younger individuals a severe trauma of the head may interfere with or arrest the personality development. The pattern of reaction may be changed because of restriction of some of the neural pathways, the presence of irritative or depressive lesions, and because of changes in the sympathetic nervous system. More frequently, in older individuals where catabolic processes are in progress and where there is limited bio-psychic plasticity, an unfavorable reaction may be noted.

B. *Post-traumatic personality disorders:* We have divided the post-traumatic personality disorders into two types:

The milder form (post-traumatic constitution) presents symptoms which are similar to those observed in encephalopathies. These cases display vasomotor changes, lack of concentration, easy fatigability, emotional instability, unreliability and irritability. The symptoms resemble those of the milder psychopathic states and psychoneuroses.

In the post-traumatic personality deviation, characterized by more severe personality and emotional changes, delusions and hallucinations as well as definite anti-social and asocial behaviour were observed. These symptoms may closely parallel those observed in manic depressive psychoses and schizophrenic states, but are never as clear-cut.

Certain difficulties may, however, arise in the differential diagnosis between traumatic psychoses and the so-called functional psychoses. The presence or absence of previous psychotic manifestations is an important point in distinguishing between the functional and traumatic psychoses. However, in those cases with psychoneurotic and psychopathic manifestations it is often difficult to decide whether one is dealing with a psychoneurosis or psychopathic state due to the head trauma, or whether the symptoms were present prior to and independent of the head trauma. The

release of previous psychoneurotic manifestations by the head trauma, which acts as a precipitating factor, has to be considered.

The criteria for a diagnosis of post-traumatic personality disorder in contrast to functional psychoses are as follows:

1. A definitely established history of a head trauma of considerable consequence associated with some degree of clouding of consciousness or unconsciousness.

2. A careful clinical and psychiatric history and neurological examination with an evaluation of the entire course.

3. The presence of personality changes following the acute and subacute manifestations mentioned above.

4. The development of delusions and hallucinations after the confusion has somewhat subsided.

5. The absence of any psychotic reactions prior to the trauma.

6. The presence of organic defect symptoms or neurologic signs and the occurrence of positive x-ray, encephalographic and spinal fluid findings as well as a positive Barany test.

In contrast to schizophrenic, traumatic personality disorders exhibit less constant and more sporadic paranoid hallucinatory episodes which are elicited in specific emotional situations. These may alternate with periods of fairly normal behavior. There is less self absorption and much more emotional display than in the true schizophrenic. Catatonic phenomena are very rarely present.

In contrast to manic psychoses, post-traumatic personality disorders exhibit a sporadic elation or irritability or dull euphoria with outbursts of emotional instability, rather than a sustained excitability. The cyclothymic features in the previous personality are not as marked. The occurrence of these symptoms following the more acute manifestations subsequent to head trauma are important considerations. In contrast to manic depressive psychoses the periodic depressions or dull apathy is not as sustained.

In regard to differentiation from psychoses with psychopathic personalities, the occurrence of the organic syndrome, sometimes during the course; the presence of more diffuse memory impairment; mental tension defects; disorders of thought, concentration and insight; more severe alteration in the personality are of importance. A careful description of the typical course following the head trauma is an aid in the diagnosis.

In differentiating the milder traumatic personality disorders, often designated as traumatic constitution, from pre-existing psychoneuroses difficulty may be encountered. The point of view of the psychiatrist is often the determinant in the evaluation of this type. However, where the typical course described above is present, where there is a history of rather severe head trauma and where the symptoms occur in intimate relation to the injury and the clinical picture, then the neurotic manifestations are the result, at least in part, of the injury and its psychological and physiological components. Moreover, in these cases designated as traumatic constitution, not only a typical history was present, but there were alterations in the personality more severe than those seen in the usual course of the psychoneuroses. Autonomic and vasomotor changes, lack of concentration, easy fatigability, dull apathy, emotional instability, unreliability and irritability, not previously present, appeared soon after the head trauma. The symptoms were not as diffuse as in the functional neurosis. Organic signs or symptoms were demonstrated at some time in the clinical course.

C. *Combined Personality and Organic Defect Type:* In this group, signs of mental deterioration and personality defects are both evident and are of relatively equal importance. The presence of the organic defect syndrome indicates that the personality changes are not of purely functional origin, and the history indicates the presence of a previously poorly integrated personality.

D. *Post-Traumatic Neuroses:* In regard to neuroses following head injuries the same considerations apply as in the cases of traumatic constitution. There are, however, fewer or no manifestations of the organic traumatic syndrome. Therefore, the greatest difficulty presents itself in distinguishing between neuroses following head injuries and endogenous psychoneuroses. While the neurosis following head injury is a more clearly defined syndrome than neurosis following somatic, peripheral or occupational trauma, some doubt the existence of this entity. There

is truth in the viewpoint that post-traumatic cranial neurotic manifestations may occur in individuals who have been more than moderately psychoneurotic. With some justification, the trauma may be regarded as playing a minor or negligible role.

When the psychoneurotic symptoms occur only after, or are markedly increased by the trauma, it does seem, however, that the head injury is a factor in the production of symptoms although not always the most important one. Even though in many cases of cephalic traumatic neurosis, the reaction is to the situation rather than to organic changes, the trauma acts as a precipitating factor in the same manner as any psychogenic or emotional trauma.

In neuroses following head injuries, the manifestations are in more direct relationship to the trauma. The symptoms are, as a rule, more circumscribed and less diffuse. The anxiety and hysterical reactions follow soon after the injury. The individual psyche concerns itself with and constantly returns to the traumatic situation, both consciously and unconsciously. Though the individual may use previous psychoneurotic patterns in reacting to the situation, these are centered about escape from the traumatic situation, the unconscious satisfaction gained from it, or the reactivation of fearful memories in regard to it. The secondary gain is concealed from the individual unless the complex in regard to the trauma is resolved or satisfied by compensation or the completion of litigation. Subsequent adaptation to various stresses, social, financial, familial and occupational, must also be considered. The trauma again may exaggerate or reactivate certain previous neurotic tendencies or complexes and must be considered as part of the constellation. If organic signs can be demonstrated at some time during the course, a diagnosis of traumatic constitution is preferable.

(4) Mixed Types

A. As we have just mentioned, some of the traumatic neuroses may be considered as combined neurotic and traumatic reactions or as neurotic states complicated by trauma. Again, in regard to personality disorders, we have previously stated that it is at times difficult to decide whether a given, unintegrated personality has developed symptoms primarily due to the head trauma, or whether a pre-existing functional psychosis, psychopathic or neurotic state is responsible for the syndrome. It seems reasonable that when these states have been previously demonstrated to exist where there is a head trauma of sufficient severity, or where confusion and delirium had been present, to consider the brain injury as a super-imposed complication. The trauma may play some role etiologically if only as an emotional factor. However, cases are frequently encountered where it can be established, with little doubt, that the head injury had nothing to do with the production of symptoms.

B. Where previous organic syndromes are present, it may often be difficult to decide which takes precedence in the clinical picture. This is particularly true in alcoholics, arteriosclerotics, seniles and cases with syphilis of the central nervous system. We have previously pointed out the occurrence of these etiological factors in combination with the head trauma. Where no psychosis was previously demonstrated, we believe that the trauma plays a more important etiological role. However, at times it is difficult to decide which is the predominating factor. In those cases where a psychosis was definitely present prior to the head trauma, while precedence must be given to the previous organic syndrome, it appears that the head trauma does play some role in the clinical picture. In those cases, particularly where there is impairment of cerebral circulation, the prognosis is poor. It is important to note that organic states, such as arteriosclerosis, may precede or occur concomitantly with or subsequent to the head trauma.

These cases may not be subjects for compensation awards according to a strictly legal interpretation. However, from the psychiatric view-point the occurrence of two psychotic syndromes superimposed or in combination in the same individual cannot be entirely disregarded.

Discussion

Considerable difference of opinion exists in regard to the chronic states following head injury. Other view-points than those discussed above must be considered.[2,3] For in-

stance, if definite localizing neurologic signs are present, the resulting personality disorder may be attributed to a purely organic factor. However, the recent observations of Browder and Myers[4] tend to cast doubt on a purely pathologic-anatomic explanation. Again, if minimal organic defect findings are demonstrable, the post-traumatic personality disorder is considered coincidental, and in no way connected with the trauma. The functional psychosis according to this viewpoint differs in no way from a schizophrenia or psychoneurosis which might have occurred independent of the trauma.

Further, distinction between traumatic personality disorders and functional states may be difficult. Where definite organic changes cannot be demonstrated; where the history concerning the head trauma is inadequate or unreliable; where there is absence of severe trauma to the head and the period of unconsciousness is transitory or ill-defined; where somatic injury is present; where there is an inadequate personality description; where details as to the subsequent course are lacking; where there is an inadequate mental status and where the presence of previous severe psychopathic or neurotic traits are demonstrable, the differential diagnosis may be unclear.

Strauss and Savitsky[5] have previously mentioned the importance of correlating the psychogenic and organic factors. Until more is known of the physiologic-chemical changes[4] in the nervous system and the total organism, a difference of opinion will exist.

Summary

In this communication, we attempted to classify traumatic mental syndromes into the acute, sub-acute and chronic types. In the chronic phases, the previous personality as well as organic findings are important. A differential diagnosis between functional states and post-traumatic personality disorders is attempted. The occurrence of mixed types, in which more than one etiologic factor may be present, is mentioned.

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PROCEEDINGS OF 1940 MEETING—CENTRAL NEUROPSYCHIATRIC ASSOCIATION

HOTEL PFISTER **October 25-26, 1940** **MILWAUKEE, WISCONSIN**

SELF INFLICTED LESIONS OF THE SKIN: Otto Foerster, M.D., Professor of Dermatology, University of Wisconsin.

This paper dealt with self-inflicted injuries of the skin which in many instances have their origin in the neuro-psychopathic states with which they are often associated. A serious medical problem may often be presented with important social and economical aspects. The salient points of this problem, especially as they relate to diagnosis, were presented.

RECENT ADVANCES IN THE CHEMOTHERAPY OF MENINGITIS: Francis R. Janney, M.D., F.A.C.P., Assistant Clinical Professor of Pediatrics, Marquette University Medical School; and Victor J. Cordes, M.D.

This included a review of the frequency of, and the mortality rates from the meningitides, particularly in children. The difficulties and effects of various types of therapy were reviewed. It was pointed out that there is urgent need for an early differential diagnosis because early and intensive chemotherapy with the right drug of the sulphanilamide series offers much toward recovery. Delayed diagnosis and inadequate dosage produce poor results. The dosage, manner of administration, management, and supervision were covered.

TREATMENT OF BULBAR AND UPPER CERVICAL TYPES OF POLIOMYELITIS BY PERIVASCULAR DRAINAGE: Max J. Fox, A.B., M.D., Associate Clinical Professor of Medicine, Marquette University Medical School, Assistant Superintendent, South View Hospital.

It was shown that the mortality rates of all types of acute poliomyelitis is not fixed but ranges from 5 per cent to 35 per cent. It is unfavorably influenced by the incidence of bulbar paralysis and the upper cervical type. The mortality in bulbar paralysis may be from 35 per cent to 50 per cent, whereas the respiratory type ranges from 20 per cent to 35 per cent.

During the epidemic of 1937 in the City of Milwaukee, nine deaths occurred in the bulbar and upper cervical types of paralysis. Because of the ineffective treatment then available for bul-

bar and upper cervical paralysis, it was planned to use saline perivascular drainage in future epidemics. The basis for adoption of this treatment followed the rationale as recommended by Retan.

In August and September 1939, 21 cases of poliomyelitis were interned at the South View Hospital. Among these there were five patients critically ill with bulbar and upper cervical involvement. These fulminating cases were treated by the intravenous injection of 0.375 per cent sodium chloride solution. This venoclysis was given over a period of approximately five hours at an interval not exceeding four hours. The five cases got well and it was further noted that the improvement in types with deglutition paralysis occurred during the course of the treatment.

TREATMENT OF ENURESIS BY THE CONDITIONING TECHNIQUE: Gilbert J. Rich, Ph.D., M.D., Director Milwaukee Guidance Clinic.

The basic problem in the treatment of enuresis is that of establishing an habitual response, namely, awakening and going to the toilet when the bladder is full. This response can be produced by a device which awakens the child at the beginning of urination. Conditioned anticipatory responses are soon attached to the original reaction to a loud sound, with the result that the patient awakens when dribbling begins, and before long, whenever the bladder is full. This type of treatment may fail to solve underlying conflicts, but it may so ease the emotional situation within the home as to make the patient and the family more accessible to psychotherapy.

EXPERIMENTS ON WAVE MECHANICS OF THE NEUROMUSCULAR SYSTEM: Eben J. Carey, M.D., Dean and Professor of Anatomy, Marquette University School of Medicine.

By means of lantern slides, Dr. Carey made a most interesting demonstration of the finer alterations in muscle striations and in the nerve endings in muscle in response to various stimuli.

OBSESSION NEUROSIS IN CHILDREN: R. A. Jefferson, M.A., M.D., Assistant Clinical Professor of Psychiatry, Marquette University School of Medicine.

In this paper were discussed the psychopathology of obsessional states in children; the etiological factors; and the methods of treatment of the child, parents, and the family milieu. Case histories as examples were presented.

EMOTIONAL FACTORS IN ALLERGIC STATES: T. L. Squier, B.S., M.D., Associate Clinical Professor of Medicine, Marquette University School of Medicine.

Nervous and psychic stimuli play an important role in allergic diseases. However, the fundamental allergic antigen-antibody reactions cannot occur from psychic stimuli alone. Emotional disturbances can precipitate allergic episodes but are secondary, and effective, only in individuals who have inherited or acquired sensitivities. The aggravating effects of psychic traumata should be recognized and reduced to a minimum, but it must be realized that if allergic equilibrium is to be achieved, the primary offenders responsible for the allergic state must be eliminated or controlled.

EXPERIMENTS TO DETERMINE THE EFFECT OF CIRCULATORY FAILURE ON SOME SIMPLE FUNCTIONS OF THE CENTRAL NERVOUS SYSTEM: Norbert Enzer, M.D., F.A.C.P., Director of Laboratories, Mt. Sinai Hospital; and E. Simonson, M.D., Research Physiologist, Mt. Sinai Hospital.

Discrepancies between the data obtained by means of the clinical methods, subjective complaints and actual working capacity are very common in heart diseases and hypertension. The reason must be that the subjective and objective state of patients does not depend only on the primary decrease of the circulatory functions, but also on the reactions of the whole organism, especially where the function of the central nervous system is deteriorated because of the lack of oxygen resulting from the circulatory insufficiency.

In 23 patients with heart disease and 45 normal subjects the fusion frequency of flicker was tested, and the degree of impairment was found to coincide with the actual state of the patient.

STUDIES ON THE GRADUAL VANISHING OF CEREBRAL BLOOD VESSELS: Percy F. Swindle, Ph.D., Professor of Physiology, Marquette University School of Medicine.

For a time during intra-uterine life each of the cerebral arteries has essentially two walls, the intima and the externa. These are seperated from one another by an intramural space filled with a jelly-like substance which later becomes invaded by some cells. The invaded jelly-like substance is commonly called the tunica media. With age and under some unknown conditions, the jelly-like substance in the arteries increases sufficiently in amount so that the lumens become either greatly narrowed or completely obliterated. The obliteration is ordinarily such a gradual process that hemorrhages may not develop or that the effects of acute infarction may not arise. During this time, however, the arterial supply to the brain is becoming less adequate for the tissue so that certain forms of unusual behavior may develop, often very gradually.

SYMPOSIUM ON SHOCK THERAPY:

Dr. Lloyd H. Ziegler, Associate Medical Director, Milwaukee Sanitarium and Dr. James A. Alston, Senior Resident, Milwaukee Sanatorium, spoke on the *Recent Developments in Shock Therapy*. They pointed out that shock therapy is not new. Techniques of inducing it may be modern but the induction of convulsions as therapy is centuries old. Since the advent of metrazol and insulin shock therapy, numerous modifications and combinations of these have appeared. Other drugs and methods have also been developed for inducing convulsions and shock-like states. These were briefly reviewed. The results of certain tests and anatomico-physiological conditions were corre-

lated with the response to shock therapy. The prognostic indicators were also reviewed.

Dr. Carroll W. Osgood, Staff Physician, Milwaukee Sanitarium spoke on *An Analysis of Disappointing Results with Metrazol in the Treatment of Certain Depressions.* He pointed out that metrazol shock therapy has come to be thought of as "specific" for depressive psychoses. Some cases, however, fail to respond as expected. An analysis was made of factors possibly responsible for such failure, and the failures contrasted with successes in other patients of like age and duration of illness.

Dr. Michael Kasak, Medical Director, Milwaukee County Hospital for Mental Diseases and Dr. Harold Schroeder, Second Year Resident at the hospital spoke on *Incidence of Relapses Following the Shock Therapies.* They pointed out the incidence of relapses of metrazol and insulin shock therapy reviewed in schizophrenia, in depressive states, drawn from their own experience and that of others. Tabulated interpretations and conclusions resulting in data were given.

Dr. Jack L. Kinsey, Staff Physician, Shorewood Hospital Sanitarium presented a paper on *The Incidence and Causes of Death Associated with Shock Therapy.* This paper was statistical in nature, dealing with the numbers and percentages of deaths in shock therapy. The causes of death were considered mainly in relation to the part of shock treatment played, but pre-existing contributory lesions were also discussed.

OBSERVATIONS ON THE TREATMENT OF AMYOTROPHIC LATERAL SCLEROSIS (LEUCOPOLIOMYELOPATHY) WITH VITAMIN E: Andrew I. Rosenberger, M.D.

This paper dealt with a report on the use of Vitamin E (Ephynal) in several cases of amotrophic lateral sclerosis and progressive muscular atrophy. The ages of the patients ranged from the early thirties to the late sixties. In most of the cases, however, the disease was far-advanced. The speaker's investigation confirms Wechsler's impression that Vitamin E is a definite therapeutic aid, not only in amyotrophic lateral sclerosis but in other allied neuro-myopathies. White rats were shown that had been on a Vitamin E deficiency diet and some foxes that had also showed neurological disturbances as the result of a diet deficiency.

VASOSPASM IN THE HANDS OF AIR HAMMER WORKERS: John L. Garvey, M.D., Clinical Professor of Neurology, Marquette University School of Medicine.

A series of cases were briefly discussed, in which the condition of spastic anemia of the fingers developed in air hammer workers. The resulting functional disability was discussed. The author had occasion to see cases reported years ago by others, and the subsequent course was commented on.

LEAD ABSORPTION, CLINICAL EFFECTS ESPECIALLY THOSE RELATING TO THE NERVOUS SYSTEM: E. L. Belknap, M.D., F.A.C.P., Assistant Clinical Professor of Medicine, Marquette University School of Medicine.

Diagnosis of lead poisoning cannot be made by any one criterion, particularly not a laboratory criterion. So-called normal lead in blood and urine was reviewed, as were high lead levels often found in lead workers who show no harmful effect. Existence of a safe and normal lead metabolism was emphasized. The term "chronic lead poisoning" was shown to be a misnomer. Distinction was drawn between non-disabling lead absorption and disabling acute lead intoxication, the real lead poisoning with its three types of lead colic, lead palsy and lead encephalopathy. Factors which transform simple lead absorption into lead intoxication were enumerated. The usual duration of temporary disability and residuals, if any, were discussed.

Diagnosis of lead poisoning must continue to rest upon the whole picture of known lead exposure with objective evidence of lead absorption as well as typical signs and symptoms of lead intoxication, all viewed in the light of clinical experience.

THE CAUDATE NUCLEUS. EFFECTS OF STIMULATION AND DESTRUCTION: David Cleveland, M.D., Ph.D., F.A.C.S.

Because of the lack of agreement as to the physiologic function of the Caudate Nucleus, an attempt is being made to add some light on the subject. The present experimental plan is limited to cats and monkeys, although other animals may be used at a later date. The Horsley-Clarke stereotaxic machine is attached to the head of the experimental animal and a fine electric needle, the point of which is bare, is inserted into the Caudate Nucleus and electrical stimulation of the Caudate Nucleus is produced by various strengths of current. In this way, the entire Caudate Nucleus is explored and the physiologic effects are carefully observed.

In a second series of animals, electrolytic destruction of the Caudate Nucleus is carried out. The lesions may vary from small, isolated ones to multiple lesions in one or both nuclei or there may be complete destruction of both Caudate Nuclei. Electrolytic destruction is carried out by the same method as in stimulation, namely, the Horsley-Clarke stereotaxic machine, which allows accurate placing of the needle in the Caudate Nucleus without destruction of any of the surrounding tissue. Experiments are in progress and no conclusions can be drawn as yet.

CHRONIC RECURRING SUBDURAL HEMATOMA IN INFANTS: M. G. Peterman, M.D.

This condition is not rare in infancy, but it is seldom recognized. There is no characteristic symptomatology. An infant suddenly has a convulsion following the subsidence of an acute

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upper respiratory infection. There may occasionally be a history of trauma. Following the convulsion, there is a progressive increase in size of the head. Usually, the convulsions increase in frequency. One case was presented in which there was no convulsion and no increase in head size. Early operation will prevent a progressive mental deterioration.

CLINICAL AND NEUROPATHOLOGICAL PRESENTATIONS: Stephen S. Stack, M.D., Assistant Professor of Pathology, Marquette University School of Medicine.

Presentation of interesting case material, including necropsy findings from such disorders as Schilder's Disease, hemangioma of nervous system, multiple myeloma with psychosis, and others, was given.